

CASE REPORT

Tachycardiomyopathy: a diagnosis not to be missed

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The prognosis of dilated cardiomyopathy is generally poor. In the vast majority of cases the cause of the ventricular dysfunction is irreversible but occasionally potentially curable causes are identified. Tachycardiomyopathy is a rare and potentially treatable cause of heart failure. A patient with a particularly severe case who had an excellent outcome is presented.

The prognosis of dilated cardiomyopathy is generally poor.¹ In the vast majority of cases the cause of the ventricular dysfunction is irreversible¹ but occasionally potentially curable causes are identified. Tachycardiomyopathy is a rare and potentially treatable cause of heart failure. We present a patient with a particularly severe case who had an excellent outcome.

CASE REPORT

An 18 year old man presented to his local hospital with a four week history of increasing shortness of breath with haemoptysis. *Streptococcus sanguis* pneumonia and severe dilated cardiomyopathy were diagnosed.

In 1994 he had undergone emergency splenectomy after trauma. There was also a history of paroxysmal supraventricular tachycardia and he had taken occasional medication sporadically. He had defaulted from follow up. Indeed there were unusual social circumstances and he had rarely been out of the house for the preceding two years.

On arrival at our unit, his heart rate was regular at 160 beats/min with a blood pressure of 80/50 mm Hg. He had signs of biventricular heart failure. ECG showed a narrow complex tachycardia at 180 beats/min with T wave inversion inferolaterally (fig 1). The P wave axis was inferior and the QRS axis rightward. Chest radiography showed cardiomegaly with an area of consolidation in the right mid-zone. Echocardiography showed globally dilated, extremely poorly

contracting ventricles (video 1: to view videos go to <http://www.heartjnl.com/supplemental>) with an estimated left ventricular ejection fraction of 8%. There was mild functional mitral and tricuspid regurgitation.

Initial management included high dose intravenous antibiotics, diuretics, inotropes, and thiamine. Results of thyroid function tests and iron studies were normal. An electrophysiology study showed no evidence of an accessory pathway. The earliest atrial activity mapped to the sinus node region. Intravenous adenosine 24 mg transiently slowed the atrial rate without a change in atrial activation or P wave morphology. This response to adenosine is characteristic of sinus rhythm.² Hence, it was concluded that the diagnosis was dilated cardiomyopathy, possibly viral in nature, with appropriate sinus tachycardia.

The patient's clinical condition deteriorated significantly over the next two weeks. He developed methicillin resistant *Staphylococcus aureus* (MRSA) pneumonia and disseminated intravascular coagulopathy with deep venous thrombi and pulmonary emboli. Treatment with heparin caused heparin induced thrombocytopenia. Over the following three weeks he slowly began to improve. Digoxin and lisinopril were successfully initiated. Repeated attempts to institute β blockers with low dose intravenous esmolol were unsuccessful because of recurrent hypotension and pulmonary oedema. Cardiac transplantation was excluded as an option because of ongoing sepsis and recent thromboembolic events.

During the period of deterioration and subsequent improvement we noted very little variation in his atrial rate between 160 and 180 beats/min. This raised the possibility of tachycardiomyopathy. We considered the alternative diagnoses of inappropriate sinus tachycardia or sinus node re-entry tachycardia induced cardiomyopathy, although these had not previously been described and we decided to proceed to a further electrophysiology study.

Repeat mapping of the sinus node region identified a focus about 1 cm inferior to the sinus node. The earliest atrial

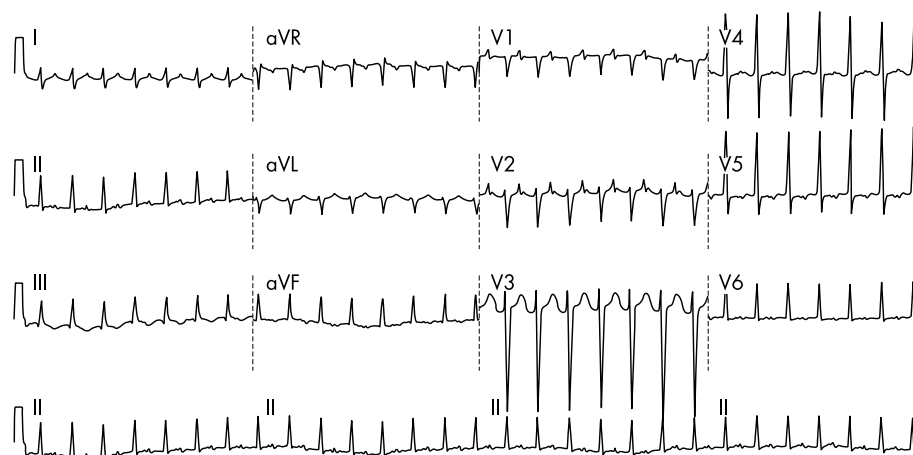


Figure 1 ECG at time of admission.

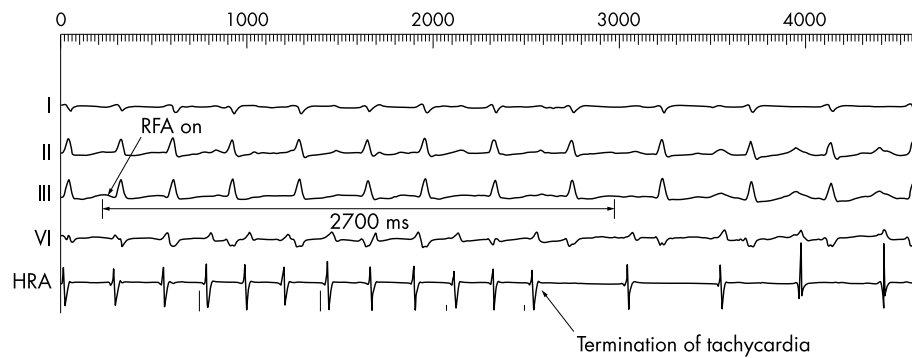


Figure 2 Abrupt fall in heart rate with radiofrequency ablation.

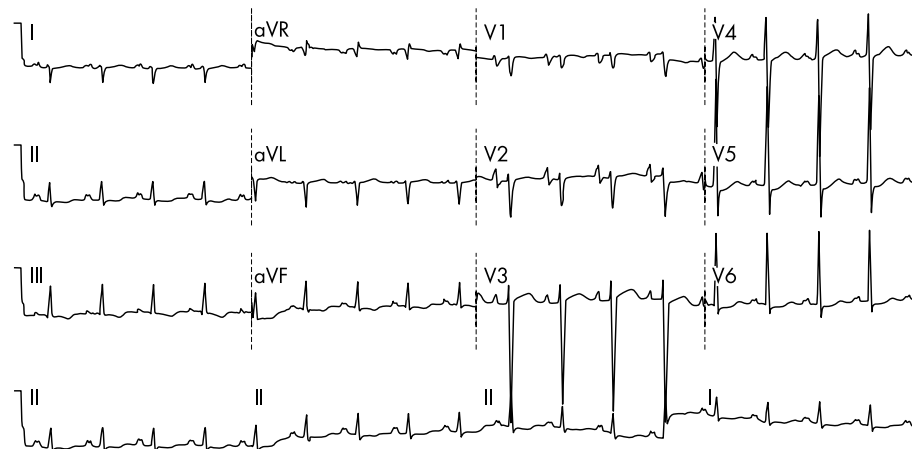


Figure 3 ECG following ablation.

activity was 58 ms before the surface P wave and radiofrequency ablation was performed at this site. There was an abrupt fall in the heart rate from 180 beats/min to 120 beats/min within four seconds of the first radiofrequency application (fig 2) and an increase in systolic blood pressure from 90 mm Hg to 120 mm Hg. The P wave morphology during sinus rhythm was almost identical to that in tachycardia (compare fig 1 with fig 3). He had no recurrent tachycardia thereafter and his clinical condition rapidly improved subjectively and objectively. The cardiomegaly as documented by chest radiography resolved rapidly, although the radiological evidence of pneumonia persisted. Oral carvedilol was successfully introduced. His discharge was delayed by a *Klebsiella* sp septicaemia. He was discharged on long term oral penicillin-V and post-splenectomy immunisations. His most recent echocardiogram nine months after ablation showed that his left ventricular end diastolic diameter had normalised to 56 mm, although there was still mild, global left ventricular dysfunction (video 2).

DISCUSSION

A review of the causes of dilated cardiomyopathy in a case series of 1278 patients found that 51% were defined as idiopathic.³ In a separate report of 673 cases of dilated cardiomyopathy only one case was attributed to incessant tachycardia.⁴ As arrhythmias are frequently the result of cardiomyopathy, they are easily overlooked as the potential cause. Atrial fibrillation, atrial flutter, ectopic atrial tachycardia, atrioventricular tachycardia, atrioventricular nodal tachycardia, and ventricular tachycardia have all been shown to cause tachycardiomyopathy.⁵

Incessant ectopic atrial tachycardia is a less common cause of tachycardiomyopathy. It accounts for 5% of cases of tachycardiomyopathy in adults and 14% in children.⁵⁻⁶

As in our case it is vital to recognise tachycardiomyopathy, as control of the tachyarrhythmia usually results in resolution of the cardiomyopathy.⁵⁻¹⁰

In summary, this 18 year old had an incessant right atrial tachycardia with resultant severe tachycardia induced cardiomyopathy. Review of the hospital records from 1994 suggested that his atrial tachycardia was misdiagnosed as sinus tachycardia and was a factor in the decision to perform splenectomy. Ablation was a curative procedure for the atrial tachycardia and resulted in dramatic improvement in his tachycardiomyopathy.

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